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<u>#61</u>	Search baculovirus and HBV Limits: Publication Date to 2000/06/09	15:00:56	<u>34</u>
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<u>#57</u>	Search AD cell line and HBV Limits: Publication Date to 2000/06/09	14:53:12	<u>20</u>
<u>#56</u>	Search AD cell line Limits: Publication Date to 2000/06/09	14:53:01	<u>5385</u>
<u>#53</u>	Search AD and HBV and compound Field: All Fields, Limits: Publication Date to 2000/06/09	14:50:34	<u>3</u>
<u>#52</u>	Search AD and HBV and compound	14:50:21	<u>4</u>
<u>#50</u>	Search Doong S 1991	14:25:43	<u>1</u>
<u>#30</u>	Search Boyd 1987 and HBV	12:03:38	<u>0</u>
<u>#49</u>	Search Kruger 1994 and HBV	12:03:11	<u>2</u>
<u>#48</u>	Search main 1996 and HBV	12:02:33	<u>32</u>
<u>#47</u>	Search Severini 1995 and HBV	12:02:20	<u>1</u>
<u>#46</u>	Search Dienstag 1995 and HBV	12:02:03	<u>2</u>
<u>#45</u>	Search Bock 1997 and HBV	12:01:43	<u>1</u>
<u>#44</u>	Search Nakbayashi 1982 and HBV	12:00:43	<u>328</u>
<u>#43</u>	Search Trautwein 1993 and HBV	12:00:20	<u>1</u>
<u>#42</u>	Search Bruss 1991 and HBV	12:00:00	<u>1</u>
<u>#41</u>	Search Briss 1991 and HBV	11:59:52	<u>0</u>
<u>#40</u>	Search Okamoto 1992 and HBV	11:59:18	<u>7</u>
<u>#39</u>	Search McMahon 1992 and HBV	11:59:03	<u>0</u>
<u>#38</u>	Search Norder 1993 and HBV	11:58:38	<u>4</u>
<u>#37</u>	Search Poch 1989 and HBV	11:58:22	<u>0</u>
<u>#36</u>	Search Poch 1989	11:58:12	<u>11</u>
<u>#35</u>	Search Bartholomeusz 1997	11:57:50	<u>3</u>
<u>#34</u>	Search Tillmann 1999 and HBV	11:57:13	<u>4</u>
<u>#33</u>	Search Tillmann 1999	11:57:07	<u>34</u>
<u>#32</u>	Search Hodge V 1993	11:56:50	<u>0</u>

<u>#31</u>	Search Hodge V 1993 and HBV	11:56:45	<u>0</u>
<u>#29</u>	Search Vere D 1993 and HBV	11:56:16	<u>0</u>
<u>#28</u>	Search Hodge 1993 and HBV	11:56:08	<u>0</u>
<u>#27</u>	Search Vere Hodge 1993 and HBV	11:56:01	<u>0</u>
<u>#26</u>	Search Summers 1982 and HBV	11:55:17	<u>7</u>
<u>#25</u>	Search Summers 1982	11:55:08	<u>74</u>
<u>#23</u>	Search Torresi J and HBV Limits: Publication Date to 2000/06/09	10:50:27	<u>1</u>
<u>#22</u>	Search Alister S and HBV Limits: Publication Date to 2000/06/09	10:50:08	<u>0</u>
<u>#20</u>	Search Trautwein C and HBV Field: All Fields , Limits: Publication Date to 2000/06/09	10:45:33	<u>14</u>
<u>#19</u>	Search Trautwein C and HBV	10:45:16	<u>28</u>
<u>#18</u>	Search Trautwein C and HBV and inhibit	10:45:05	<u>2</u>
<u>#16</u>	Search Manns M and HBV treatment	10:44:32	<u>32</u>
<u>#17</u>	Search Manns M and HBV and inhibit	10:43:59	<u>2</u>
<u>#15</u>	Search Manns M and HBV	10:43:29	<u>64</u>
<u>#14</u>	Search Tillmann H and HBV	10:43:00	<u>30</u>
<u>#12</u>	Search Bock T and HBV	10:40:43	<u>7</u>
<u>#11</u>	Search Bock T	10:40:36	<u>72</u>
<u>#6</u>	Search anti-HBV and cell line and screening	10:06:07	<u>4</u>
<u>#5</u>	Search anti-HBV and cell line	10:05:59	<u>59</u>
<u>#2</u>	Search anti-HBV and detect	09:53:17	<u>4</u>
<u>#1</u>	Search anti-HBV drug	09:52:34	<u>147</u>

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May 12 2004 06:43:00

=> HBV
L1 19429 HBV

=> screening (w) assay
L2 4580 SCREENING (W) ASSAY

=> L1 and L2
L3 24 L1 AND L2

=> inhibit (l) L1
L4 637 INHIBIT (L) L1

=> assay
L5 808989 ASSAY

=> L4 and L5
L6 125 L4 AND L5

=> cell (l) line
L7 692543 CELL (L) LINE

=> L1 and L7
L8 1693 L1 AND L7

=> L8 and L6
L9 47 L8 AND L6

=> vector or plasmid
L10 503456 VECTOR OR PLASMID

=> L10 and L1
L11 2220 L10 AND L1

=> L5 and L11
L12 302 L5 AND L11

=> L12 and L9
L13 10 L12 AND L9

=> D L10 IBIB ABS 1-10

=> "drug screening"
L14 27763 "DRUG SCREENING"

=> L1 and L14
L15 57 L1 AND L14

=> inibit and L15
L16 0 INIBIT AND L15

=> inhibit and L15
L17 13 INHIBIT AND L15

=> D L17 IBIB ABS 1-13

L17 ANSWER 1 OF 13 CAP

L13 ANSWER 8 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:402599 BIOSIS
DOCUMENT NUMBER: PREV199900402599
TITLE: Specific inhibition of hepatitis B virus replication by
sense RNA.
AUTHOR(S): Putlitz, Jasper Zu; Wands, Jack R. [Reprint author]
CORPORATE SOURCE: Liver Research Center, Rhode Island Hospital, Brown
University School of Medicine, 55 Claverick Street,
Providence, RI, 02903, USA
SOURCE: Antisense and Nucleic Acid Drug Development, (June, 1999)
Vol. 9, No. 3, pp. 241-252. print.
ISSN: 1087-2906.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 8 Oct 1999
Last Updated on STN: 8 Oct 1999

AB We describe effects of sense RNA molecules on hepatitis B virus (HBV) replication and antigen synthesis in transiently transfected cells. When certain subgenomic fragments of HBV were expressed as sense RNA together with a replication-competent genome of HBV, they inhibited HBV replication by up to 75% and HBsAg secretion by up to 60%. The corresponding antisense sequences had a 50% inhibitory effect in one case and no effect in another case. The sense RNA species did not inhibit duck hepatitis B virus (DHBV) replication, suggesting specific inhibitory effects. HBV transcript levels were unaltered in the presence of sense RNA species, consistent with an inhibitory effect mediated at the posttranscriptional level. The inhibition of HBV replication by overexpression of sense RNA derived from the viral genome represents an example of sense cosuppression of an animal virus.

L13 ANSWER 7 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:330563 BIOSIS
DOCUMENT NUMBER: PREV200300330563
TITLE: Construction of hepatocellular specific preS2 antisense RNA
expression **vector** and study of its inhibitory
effect.
AUTHOR(S): Ma Chun-hong; Sun Wen-sheng [Reprint Author]; Zhang
Li-ning; Cao Ying-Lin; Wang Zhen-guang; Liu Su-xia; Wang
Xiao-Yan; Zhu Fa-liang
CORPORATE SOURCE: Medical School, Institute of Immunology, Shandong
University, Ji-nan, 250012, China
sunwenshengsws@yahoo.com
SOURCE: Zhonghua Weishengwuxue He Mianyixue Zazhi, (April 2003)
Vol. 23, No. 4, pp. 279-282. print.
CODEN: ZWMZDP. ISSN: 0254-5101.
DOCUMENT TYPE: Article
LANGUAGE: Chinese
ENTRY DATE: Entered STN: 16 Jul 2003
Last Updated on STN: 16 Jul 2003

AB Objective: To obtain recombinant preS2 antisense RNA expression
vector targeted to hepatocellular carcinoma. Methods: preS2 gene
(3203-340) was obtained by PCR and then cloned into EB virus
vector pEBAF containing human alpha-fetoprotein promoter and
enhancer. After transfection by lipofectin, RNA dot blot, ELISA and
real-time qualitative PCR were used to **assay** the expression
preS2 mRNA, **HBV** antigens and **HBV** DNA respectively.
Results: The recombinant pEBAF-as-preS2 which can be expressed
specifically in hepatocellular carcinoma **cells** was successfully
obtained. Both **HBV** DNA and **HBV** antigens of
hepatocellular **cell line** HepG2.2.15 were inhibited by
transfection by pEBAF-as-preS2. The inhibition rate of HBsAg, HBeAg and
HBV DNA were 33.4%, 41.5% and 86% respectively. Conclusion:
pEBAF-as-preS2 expression **vector** may lead to targeted-expression
of preS2 antisense RNA on hepatoma **cells** and **inhibit**
HBV multiplication.

L13 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1998:473074 CAPLUS

DOCUMENT NUMBER: 129:271193

TITLE: Inhibition of hepatitis B virus by retroviral
vectors expressing antisense pre-C/C gene
fragment

AUTHOR(S): Ji, Wei; Wang, Qinhuang; Si, Chongwen; Zhang, Guoqing

CORPORATE SOURCE: The Fourth Department of Infectious Diseases, The
302nd Hospital of the PLA, Beijing, 100039, Peop. Rep.
China

SOURCE: Zhongguo Bingduxue (1997), 12(3), 214-218

CODEN: ZBINER; ISSN: 1003-5125

PUBLISHER: Kexue Chubanshe

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB Retroviral **vector** expressing antisense RNA complementary to
hepatitis B virus (HBV) DNA pre-C/C region (ANTI-C) was used to
transduce the human hepatoblastoma **cell line** 2.2.15,
which was transfected with HBV DNA and can express HBV
markers. The inhibitory effects of antisense gene mediated by retroviral
vector ANTI-C on the expression of HBV antigens appeared
as early as on the 3rd day after transduction, reached peak level on the
5th day, and persisted at least for 11 days. The inhibitory rates of
HBsAg and HBeAg expression in 2.2.15 **cells** by ANTI-C were 27.0%
and 59.5% on the 5th day after transduction. HBV DNA in the
supernatant of 2.2.15 **cells** transduced with ANTI-C also reduced
on the 5th day after transduction as detected by DNA dot blot
assays. The viability of transduced 2.2.15 **cells** was
not affected. Antisense retroviral **vectors** containing antisense
gene can **inhibit** HBV and may be potentially useful for
anti-HBV gene therapy.

L13 ANSWER 6 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 2004:134204 BIOSIS

DOCUMENT NUMBER: PREV200400132270

TITLE: LY582563 (MCC-478), a 2-aminopurine nucleotide analogue,
inhibits packaging and replication of HBV
genome in vitro.

AUTHOR(S): Shaw, Tim [Reprint Author]; Colledge, Danni [Reprint
Author]; Sozzi, Vitina [Reprint Author]; Locarnini, Stephen
A. [Reprint Author]

CORPORATE SOURCE: Victorian Infectious Diseases Reference Laboratory, North
Melbourne, VIC, Australia

SOURCE: Hepatology, (October 2003) Vol. 38, No. 4 Suppl. 1, pp.
716A. print.

Meeting Info.: 54th Annual Meeting of the American
Association for the Study of Liver Diseases. Boston, MA,
USA. October 24-28, 2003. American Association for the
Study of Liver Diseases.

ISSN: 0270-9139 (ISSN print).

DOCUMENT TYPE: Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 10 Mar 2004

Last Updated on STN: 10 Mar 2004

AB Background: LY582563, a new synthetic nucleotide analogue developed
jointly by Mitsubishi Pharmaceutical Company and Eli Lilly, is a potent
inhibitor of replication of both wild type (wt) and lamivudine (LMV)
resistant HBV strains. Prior experiments indicate that its
mechanism(s) of action differs from those of LMV and adefovir. Aims: To
investigate the mechanisms by which LY582563 **inhibits**
HBV replication, in vitro. Methods: Compound LY58263 was provided
by Eli Lilly. The following viral parameters were investigated: (1)
Endogenous HBV polymerase activity was measured in **cell**

free **assays** using isolated, partly purified virus particles as the enzyme source. (2) Biosynthesis of viral antigens and replicative intermediates including **HBV** ccc DNA were studied in HepG2 **cells** transduced with wt **HBV** using a recombinant baculovirus **vector**. (3) Three derivatives of the Huh-7 **cell line**, in which the expression of **HBV** core protein is controlled by a tetracycline responsive element (pTRE) were used to study packaging in trans. These were: pTRE (control), PC47 (precore producing; no rescue control), C4B (core producing; will rescue). (4) AD-38 HepG2 **cells**, in which expression of an integrated **HBV** genome occurs in response to tetracycline withdrawal, were used to compare the antiviral effects of LY582563 and interferon-alpha (IFN). Production of **HBV** RNA, intra- and extra- cellular DNA and nuclear ccc DNA was monitored by Northern blotting, quantitative RT-PCR (QPCR), and Southern blotting respectively. Viral antigens were detected and quantified by SDS-PAGE and immunoblotting. Southern blotting, QPCR, immunoblotting and electron microscopy (EM) were used to study capsid formation and encapsidation of pregenomic RNA (pgRNA). Results: Phosphorylated metabolites of LY582563 did not **inhibit** endogenous **HBV** DNA polymerase activity. LY582563 had no significant effect on generation of **HBV** cccDNA, RNA, or the expression of viral antigens (HBe, HBs or HBc) in the recombinant baculovirus system. Inhibition of packaging of the pgRNA in cis, but not in trans, was observed in both baculovirus and pTRE based **assays**. EM examination revealed that the effect on packaging was not due to inhibition of core dimerisation or production of nucleocapsids. Furthermore, the antiviral effect of LY582563 in AD38 HepG2 **cells** did not appear to be mediated by an IFN-like mechanism. Conclusions: LY582563 interfered with the packaging of **HBV** pgRNA into nucleocapsids. Since correctly assembled nucleocapsids are a prerequisite for efficient **HBV** replication, interference with the packaging of **HBV** pgRNA by LY582563 may be the primary mechanism by which it **inhibits** **HBV** replication. Further studies are required to determine whether LY582563 specifically or differentially **inhibits** priming of reverse transcription or RNase H activity. Based on this novel mechanism of action and its activity against wt and LMV-resistant **HBV**, further clinical studies of LY582563 appear warranted.

L13 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1999:185369 CAPLUS

DOCUMENT NUMBER: 131:17201

TITLE: Hepatitis B virus X protein inhibits nucleotide excision repair

AUTHOR(S): Jia, Libin; Wang, Xin Wei; Harris, Curtis C.

CORPORATE SOURCE: Laboratory of Human Carcinogenesis, Division of Basic Sciences, National Institutes of Health, Bethesda, MD, USA

SOURCE: International Journal of Cancer (1999), 80(6), 875-879

CODEN: IJCNAW; ISSN: 0020-7136

PUBLISHER: Wiley-Liss, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Human hepatitis B virus (HBV) is a major risk factor of human hepatocellular carcinoma. Both in vivo and in vitro studies have shown that HBV X protein (HBx) can bind to the p53 tumor-suppressor protein and interfere with the role that p53 plays in the cellular response to DNA damage. Our previous work has shown that HBx protein **inhibits** p53 sequence-specific transcriptional activation, p53-mediated apoptosis and p53 binding to the TFIIH transcription-nucleotide excision repair (NER) factors, including XPB and XPD. To investigate whether HBx interferes with the NER pathway, we utilized **cell-proliferation and colony-formation assays** to determine if **cells** expressing HBx are more sensitive to UVC-induced DNA damage. NER was also measured by a **plasmid host cell re-activation assay** using a **vector** containing a luciferase reporter gene. UV-irradiated **plasmids** were transfected into a human RKO colon carcinoma **cell line** that contains wild-type (wt) p53 as well as its derivs., either mutant p53-143ala (RKO-143ala) or human papillomavirus E6 (RKO-E6, a wt p53 protein that is rapidly degraded and non-functional). We found that **cells** expressing HBx are more sensitive to UVC-induced killing. Moreover, expression of HBx resulted in a reduction of NER efficiency in RKO **cells** to 52±2% (compared with control), RKO-143ala **cells** to 46±3% and RKO-E6 **cells** to 60±3%. Similar results were also obtained with a HepG2 hepatoblastoma **cell line** carrying wt p53. In addition, we found that HBx bound directly to either XPB or XPD DNA helicase in vitro. Thus, our data indicate that HBx may interfere with the NER pathway through both p53-dependent and p53-independent mechanisms. Because HBx binds to TFIIH-associated proteins, we propose that HBx may interfere with the NER pathway also through binding to and altering the activities of helicases necessary for NER and, thereby, increase the mutation rate induced by chemical carcinogens, such as aflatoxin B1, during human liver carcinogenesis.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ACCESSION NUMBER: 2003:703627 CAPLUS
DOCUMENT NUMBER: 140:179972
TITLE: Inhibition of **HBV** by ribozyme in adenovirus
expression system
AUTHOR(S): Li, Jinge; Zhang, Rong; Zhou, Yongxing; Lian, Jianqi;
Wang, Jiuping
CORPORATE SOURCE: Tangdu Hospital, Fourth Military Medical University,
Xi'an, 710032, Peop. Rep. China
SOURCE: Jiefangjun Yixue Zazhi (2003), 28(3), 243-245
CODEN: CFCHBN; ISSN: 0577-7402
PUBLISHER: Jenminjun Chubanshe
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB The inhibition of **HBV** expression by multi-target
ribozyme-expressing adenovirus **vector** was studied in vivo. The
ribozyme and muta-ribozyme were cloned into adenovirus **vector**
pAd-CMV. By using the adenovirus expression system, the two
plasmids were co-transfected into 293 **cell line**
with adenovirus gene recombinant **plasmids** pJM17. The
recombinant adenovirus were detected by plaque **assay**. Then the
2.2.15 **cell** were infected with recombinant adenovirus. Using
ELISA, dot-bolt hybridization and image anal. system, the inhibition of
HBV expression was detected. The ribozyme and muta-ribozyme were
cloned into the adenovirus **vector** pAd-CMV. The recombinant
adenovirus could infect 2.2.15 **cells** and express the ribozyme.
The ribozyme could decrease the expression of HBeAg by 87.1%. These
results indicate that ribozyme can **inhibit HBV**
expression in the **cell**, which represents a potential gene
therapy for **HBV** infection.

L17 ANSWER 13 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1998:592171 CAPLUS

DOCUMENT NUMBER: 130:10292

TITLE: Study on screening anti-HBV agents from antisense oligodeoxynucleotide and their toxicity

AUTHOR(S): Han, Jinxiang; Wang, Yulin; Zhang, Cui

CORPORATE SOURCE: Shandong Medicinal Biotechnology Centre, Jinan, 250062, Peop. Rep. China

SOURCE: Zhongguo Shenghua Yaowu Zazhi (1998), 19(3), 131-135
CODEN: ZSYZFP; ISSN: 1005-1678

PUBLISHER: Zhongguo Shenghua Yaowu Zazhi Bianjibu

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB To screen a new anti-HBV agent, according to sequence of HBV genome, 5 complementary sites were selected and the antisense phosphorothioate oligodeoxynucleotides (s-asODN) were designed and synthesized, they are S gene initiator, SP II cap site, Pre-C gene initiator, C gene initiator and gene ϵ initiator. The complementarity to different HBV genome were assayed for their anti-HBV activity in HepG22215 cell line with ELISA method, and the toxicity of s-asODN also was studied with MTT colorimetric assay and electron microscope. The result shows that s-asODN can inhibit the expression of HBsAg and HBeAg, and the inhibition shows sequence-specific, dose-related, antinuclease and combination-cooperative, and harmless to host characteristic in the viability, the shape and ultrastructure of cells. The study had laid the foundation of exploring new anti-HBV agents.

ANSWER 10 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
 ACCESSION NUMBER: 2000:227816 CAPLUS
 DOCUMENT NUMBER: 132:261362
 TITLE: A quick in vitro assay for viral DNA polymerase activity of hepatitis B virus and its use in antiviral drug screening
 INVENTOR(S): Oon, Chong Jin; Chen, Wei Ning; Lim, Gek Keow; Leong, Ai Lin
 PATENT ASSIGNEE(S): Government of the Republic of Singapore, Singapore
 SOURCE: PCT Int. Appl., 24 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000018958	A2	20000406	WO 1998-SG77	19980929
W:			AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM	
RW:			GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG	
AU 9893726	A1	20000417	AU 1998-93726	19980929
CA 2344282	AA	20000406	CA 1999-2344282	19990313
WO 2000018968	A1	20000406	WO 1999-SG17	19990313
WO 2000018968	C1	20021107		
W:			AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM	
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AU 9929694	A1	20000417	AU 1999-29694	19990313
TR 200100899	T2	20010723	TR 2001-200100899	19990313
EP 1117843	A1	20010725	EP 1999-910940	19990313
R:			AT, BE, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE, PT, IE, FI	
BR 9914099	A	20010731	BR 1999-14099	19990313
JP 2002525130	T2	20020813	JP 2000-572415	19990313
NZ 510704	A	20021025	NZ 1999-510704	19990313
RU 2202620	C2	20030420	RU 2001-108496	19990313
NO 2001001510	A	20010528	NO 2001-1510	20010323
HR 2001000230	A1	20020630	HR 2001-230	20010327
US 6593082	B1	20030715	US 2001-674465	20010618
US 2003082529	A1	20030501	US 2002-231405	20020828

PRIORITY APPLN. INFO.:
 WO 1998-SG77 A 19980929
 WO 1999-SG17 W 19990313
 US 2001-674465 A3 20010618

AB The hepatitis B virus (HBV) DNA polymerase for the assay is obtained from coupled in vitro transcription/translation of PCR products of serum derived HBV genomic DNA using primers containing SP6 RNA polymerase promoter sequence. The HBV DNA polymerase activity is measured by filtration assay for the priming activity in the presence of a radio-labeled nucleotide. The assay is tested with two HBV escaping mutants containing mutation in 'a' epitope of HBsAg (not protected by the vaccines, 's' 126 or 's' 145) and also used for screening for antiviral drugs that inhibit HBV DNA polymerase.

L17 ANSWER 9 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:368224 CAPLUS
DOCUMENT NUMBER: 136:83781
TITLE: Hepatitis B virus transgenic mice for model of
anti-hepatitis B virus drug study
AUTHOR(S): Xiong, Yili; Jia, Yanzheng; Wang, Hongmin; Liu,
Guangze; Ren, Hong; Zhou, Zhi; Zhang, Dingfeng
CORPORATE SOURCE: Transgenic Engineering Lab, infectious Disease Center
of PLA, 458th Hospital of PLA, Canton, 510602, Peop.
Rep. China
SOURCE: Zhonghua Ganzangbing Zazhi (2001), 9(1), 19-21
CODEN: ZGZZFE; ISSN: 1007-3418
PUBLISHER: Chongqing Yike Daxue, Dier Linchuang Xueyuan
DOCUMENT TYPE: Journal
LANGUAGE: Chinese

AB The hepatitis B virus (HBV) transgenic mice models was
established for studying if the model can be used for the evaluation of
anti-HBV drugs. HBV transgenic mice models were
produced by microinjection to analyze the integration, expression of
HBV in the transgenic mice by nested PCR, southern blot,
immunohistochem., and ELISA. Sixty mice whose HBV DNA, HBsAg
were pos. were divided into 6 groups randomly, in which 3 groups were
given drugs: lamivudine administered by perfusion of stomach tube (100
mg.kg⁻¹. day⁻¹ for 21 days); thymosin administered by abdomen injection
(3 mg.day⁻¹ for 90 days); and DNA vaccine of 100 μ g by muscle
injection. The other 3 groups were neg. control. Lamivudine, thymosin
and DNA vaccine made HBV DNA become neg. in the serum of
HBV transgenic mice. The neg. ratio was highest in lamivudine
treatment group. HBV DNA became pos. again when lamivudine
terminated. Lamivudine, thymosin, and DNA vaccine can inhibit
HBV replication. Transgenic mice might be used as the model for
anti-HBV drug screening and eval

L17 ANSWER 8 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2001:798297 CAPLUS

DOCUMENT NUMBER: 135:352761

TITLE: Synthetic peptides that bind to the hepatitis B virus core and e antigens

INVENTOR(S): Sallberg, Matti

PATENT ASSIGNEE(S): Tripep AB, Swed.

SOURCE: PCT Int. Appl., 82 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 7

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001081421	A2	20011101	WO 2001-IB844	20010420
WO 2001081421	A3	20020404		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MY, NZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6417324	B1	20020709	US 2000-556605	20000421
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PRIORITY APPLN. INFO.: US 2000-556605 A 20000421

AB The present invention relates generally to the field of virol. More particularly, the invention relates to the discovery that peptides, which bind to the Hepatitis B virus (HBV) core and e antigens, can be used to inhibit HBV infection. Embodiments concern "binding partners," which include peptides, peptidomimetics, and chems. that resemble these mols. that interact with HBV core and e antigens, biol. complexes having HBV core and e antigens joined to said binding partners, methods of identifying such binding partners, pharmaceuticals having binding partners, and methods of treatments and prevention of HBV infectio

L17 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2002:458401 CAPLUS

DOCUMENT NUMBER: 138:100375

TITLE: Characterizing antiviral activity of adefovir dipivoxil in transgenic mice expressing hepatitis B virus

AUTHOR(S): Julander, Justin G.; Sidwell, Robert W.; Morrey, John D.

CORPORATE SOURCE: Animal, Dairy, and Veterinary Sciences Department, Institute of Antiviral Research, Utah State University, Logan, UT, 84322-4700, USA

SOURCE: Antiviral Research (2002), 55(1), 27-40
CODEN: ARSRDR; ISSN: 0166-3542

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Oral adefovir dipivoxil (ADV) reduced viral load in transgenic mice expressing hepatitis B virus (HBV). Liver HBV DNA was reduced to <0.1 pg of viral DNA per μg of total DNA ($\text{pg}/\mu\text{g}$) following oral ADV therapy at a dosage of 100 mg/kg/day twice daily for 10 days as compared to a mean of 3.0 $\text{pg}/\mu\text{g}$ for the placebo control group. Oral ADV treatment also reduced serum HBV DNA to 3.5 log₁₀ genomic equivalent (ge)/mL compared to 5.3 log₁₀ ge/mL for the placebo control group. With once daily treatments, ADV antiviral activity reached near maximum viral reduction by day 10 in the liver and reached an endpoint of liver virus inhibition at 1.0 mg/kg/day. The min. ED was less than 0.1 mg/kg/day using inhibition of serum virus. Lamivudine (3TC) given orally at 500 mg/kg/day using the same treatment schedule marginally reduced the serum HBV DNA by 4-fold, but did not significantly reduce HBV liver DNA. Serum titer reduction was also identified in untreated or placebo-treated animals, which may have been caused by the stress of pre-treatment bleeding and multiple oral gavage treatments. This trauma/placebo-effect may have masked the extent of viral reduction in the serum in ADV- and 3TC-treated animals. Liver HBV RNA was not reduced by oral ADV treatments. The lack of RNA reduction was expected, because the HBV transgene is stably integrated into the chromosome and ADV inhibits polymerase activity after transcription of pregenomic RNA. ADV was identified to have potent anti-HBV activity in this HBV transgenic mouse model and could serve as a suitable pos. control for future drug discovery expts.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

DOCUMENT NUMBER: 137:348398
 TITLE: Recombinant expression of human **HBV** reverse transcriptase (RT) for *Phyllanthus amarus*-derived antiviral **drug screening**
 INVENTOR(S): Buchner, Johannes; Muschler, Paul; Halsbeck, Martin
 PATENT ASSIGNEE(S): Phytrix A.-G., Germany
 SOURCE: PCT Int. Appl., 39 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002088343	A2	20021107	WO 2002-EP4642	20020426
WO 2002088343	A3	20030424		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
EP 1390481	A2	20040225	EP 2002-740526	20020426
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR			

PRIORITY APPLN. INFO.: EP 2001-110073 A 20010427
 WO 2002-EP4642 W 20020426

AB The present invention relates to a method of producing a functional cell-free hepatitis B virus (**HBV**) reverse transcriptase (RT) comprising the steps of expressing **HBV**-RT in *E. coli* cells employing a suitable expression plasmid, or in a cell-free transcription-translation system and if expression was carried out in *E. coli* cells, lysing said *E. coli* cells and purifying **HBV**-RT from *E. coli* lysate or from said transcription-translation system. The invention further relates to a method of screening for an inhibitor of **HBV**-RT activity comprising the steps of contacting the cell-free **HBV**-RT produced according to methods of the present invention and with a potential inhibitor and assaying whether said potential inhibitor **inhibits** **HBV**-RT activity. Furthermore, the invention provides for a method of producing a pharmaceutical composition comprising the step of formulating the inhibitor identified by the screening method of the invention into a pharmaceutical compn

L17 ANSWER 4 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
 ACCESSION NUMBER: 2002:869219 CAPLUS
 DOCUMENT NUMBER: 137:363028
 TITLE: **Drug screening** assays and kits for
 discovery of anti-microbial and chemotherapeutics
 agents
 INVENTOR(S): McCarthy, Lawrence; Kong, Lilly; Shao, Tang; Su, Xin
 PATENT ASSIGNEE(S): Focus Technologies, Inc., USA
 SOURCE: PCT Int. Appl., 94 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002090993	A2	20021114	WO 2001-US44783	20011127
WO 2002090993	A3	20040415		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003039957	A1	20030227	US 2001-996187	20011127
PRIORITY APPLN. INFO.:				
			US 2000-253150P	P 20001127
			US 2001-304533P	P 20010709
			US 2001-297686P	P 20010712
			US 2001-996187	A2 20011127

AB Methods and compns. for detecting the phenotype of a bioactive mol.
 assays. More specifically, are provided methods and compns. are provided
 for determining the suitability of one ore more candidate compds. prior to or
 during the course of chemotherapy or anti-infective therapy, for their
 capacity to **inhibit** the bioactive mols. of micro-organisms,
 cancers and as an assay for expression in transgene therapy. Also
 provided are phenotypic assays for drug discovery. Claimed sequences were
 not present at the time of publication.

L17 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2004 ACS on STN
 ACCESSION NUMBER: 2002:368930 CAPLUS
 DOCUMENT NUMBER: 136:374804
 TITLE: Synthetic peptides that bind to the hepatitis B virus
 core and e antigens
 INVENTOR(S): Sallberg, Matti
 PATENT ASSIGNEE(S): Swed.
 SOURCE: U.S. Pat. Appl. Publ., 56 pp., Cont.-in-part of U.S.
 Ser. No. 556,605.
 CODEN: USXXCO
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 7
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002058247	A1	20020516	US 2001-839447	20010420
US 6417324	B1	20020709	US 2000-556605	20000421
US 2003235815	A1	20031225	US 2003-369060	20030214
US 2003225251	A1	20031204	US 2003-372735	20030221
PRIORITY APPLN. INFO.:			US 2000-556605	A2 20000421
			WO 1995-SE468	W 19950427
			US 1996-737085	A1 19961227
			US 1999-246258	A1 19990208
			US 2000-532106	A1 20000321
			US 2000-664025	A2 20000919
			US 2000-664945	A2 20000919
			US 2001-839666	A1 20010419
			US 2001-839447	B1 20010420
			WO 2001-IB2327	A2 20010919
			US 2002-153271	A2 20020521
			US 2002-234579	A2 20020830

AB The present invention relates generally to the field of virol. More particularly, the invention relates to the discovery that peptides, which bind to the Hepatitis B virus (HBV) core and e antigens, can be used to inhibit HBV infection. Embodiments concern "binding partners", which include peptides, peptidomimetics, and chems. that resemble these mols. that interact with HBV core and e antigens, biol. complexes having HBV core and e antigens joined to said binding partners, methods of identifying such binding partners, pharmaceuticals having binding partners, and methods of treatments and prevention of HBV infectio

L17 ANSWER 12 OF 13 - CAPLUS - COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1999:398699 CAPLUS

DOCUMENT NUMBER: 131:165030

TITLE: Combinatorial screening and intracellular antiviral activity of hairpin ribozymes directed against hepatitis B virus

AUTHOR(S): Putlitz, Jasper zu; Yu, Qiao; Burke, John M.; Wands, Jack R.

CORPORATE SOURCE: Molecular Hepatology Laboratory, Massachusetts General Hospital Cancer Center, Harvard Medical School, Boston, MA, 02129, USA

SOURCE: Journal of Virology (1999), 73(7), 5381-5387
CODEN: JOVIAM; ISSN: 0022-538X

PUBLISHER: American Society for Microbiology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB A combinatorial screening method has been used to identify hairpin ribozymes that inhibit hepatitis B virus (HBV) replication in transfected human hepatocellular carcinoma (HCC) cells. A hairpin ribozyme library (5+105 variants) containing a randomized substrate-binding domain was used to identify accessible target sites within 3.3 kb of full-length in vitro-transcribed HBV pregenomic RNA. Forty potential target sites were found within the HBV pregenomic RNA, and 17 sites conserved in all four subtypes of HBV were chosen for intracellular inhibition expts. Polymerase II and III promoter expression constructs for corresponding hairpin ribozymes were generated and cotransfected into HCC cells together with a replication-competent dimer of HBV DNA. Four ribozymes inhibited HBV replication by 80, 69, 66, and 49%, resp., while catalytically inactive mutant forms of these ribozymes affected HBV replication by 36, 28, 0, and 0%. These findings indicate that the inhibitory effects on HBV replication were largely mediated by the catalytic activity of the ribozymes. In conclusion, the authors have identified catalytically active RNAs by combinatorial screening that mediate intracellular antiviral effects on HBV.

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT